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EXAMINER	
KOLKER, DANIEL E	

ART UNIT	PAPER NUMBER
1649	

DATE MAILED: 06/01/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/807,414	EISENBACH-SCHWARTZ ET AL.	
	Examiner	Art Unit	
	Daniel Kolker	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *20 March 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-71* is/are pending in the application.

4a) Of the above claim(s) *5,7-9,11-20,24,27-39,43 and 47-71* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,6,10,21-23,25,26,40-42 and 44-46* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☒ Claim(s) *1-71* are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *3/24/04,8/3/04*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. Applicant's remarks filed 20 March 2006 have been entered. Claims 1 – 71 are pending.

Election/Restrictions

2. Applicant's election with traverse of Group I, claims 1 – 58, and "stroke" as the specific disease in the reply filed on 20 March 2006 is acknowledged. The traversal is on the ground(s) that restriction between the plethora of diseases and conditions listed in the claims is improper. This is not found persuasive for the following reasons:

A) Applicant argues, on p. 2 of the remarks, that there is a common mechanism for treatment of all the recited diseases, namely administration of the same compound, poly Glu,Tyr. However the diseases do not share a common mechanism or common patient populations. For example, age-related macular degeneration, recited in claim 8, has nothing to do with alcoholic neuropathy, recited in claim 32. The former is associated with advanced age and search for methods of treating the disease would necessarily be limited to articles and patents discussing age-related changes in the eyes. On the other hand, search for methods of treatment of alcoholic neuropathy would be focused on neural cell death within the brain, and would involve a detailed search of the mechanisms and causes of said neuropathy. The diseases recited in the claims comprise diseases of cardiac tissue, damage to peripheral nerves which are known to regenerate spontaneously, as well as damage to neurons of the central nervous system which do not show such spontaneous regrowth. There is not art of record evidencing a common mechanism of all the recited diseases and conditions. For example, myocardial infarcation, recited in claim 23, is commonly known as a heart attack and involves decreased blood flow to cardiac tissue. It is not a neuronal disease or condition, or a neurological injury. At this time the generic claims are not allowable and do not link all recited diseases and conditions. As the diseases listed throughout the claims share no obvious common features, other than the fact that those who have them undergo a certain amount of suffering, restriction between them is proper for search and examination purposes.

B) Applicant also argues, on p. 5 of the remarks, that in US Patent 6,844,314 the examiner accepted a common mechanism for COP-1 and thus in the instant case the examiner should consider all the disease, which share no common mechanism, together. This is not persuasive because each application is examined on its own merits; whether or not a different examiner failed to implement the same restriction requirement in a different case is immaterial.

Second, from reading the prosecution history of the '314 patent it is clear that a requirement for election of species between nerve regeneration in the PNS and CNS was made and importantly the requirement to elect a species was not traversed (see applicant's remarks filed 2 January 2003, p. 7, in application serial number 09/765544). Thus relying on the prosecution history of the '314 patent appears to support the examiner's position that requiring restriction is in fact proper.

The requirement is still deemed proper and is therefore made FINAL.

3. On p. 3 of the restriction requirement, the examiner indicated that applicant was required to indicate which claims read on the elected invention. However, no such list was provided in the reply filed 20 March 2006. The examiner has determined that the following claims either recite or encompass stroke: 1 – 4, 6, 10, 21 – 23, 25 – 26, 40 – 42, and 44 – 46.

Claims 5, 7 – 9, 11 – 20, 24, 27 – 39, 43, and 47 – 71 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 20 March 2006.

Claims 1 – 4, 6, 10, 21 – 23, 25 – 26, 40 – 42, and 44 – 46 are under examination.

Claim Objections

4. Claims 4, 23, and 42 objected to because of the following informalities: they recite non-elected subject matter, specifically diseases other than stroke. Appropriate correction is required.

Claim Rejections - 35 USC § 112

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1 – 4, 6, 10, 21 – 23, 25 – 26, 40 – 42, and 44 – 46 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for reduction of the size of ischemia-induced neural damage and decreasing the amount of neuronal cell loss within the retina by administration of poly-Glu,Tyr, does not reasonably provide enablement for

promoting nerve regeneration, or preventing neuronal degeneration, or for amounts effective to prevent, inhibit, or promote nerve regeneration. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

There are many factors considered when determining if the disclosure satisfies the enablement requirement and whether any necessary experimentation is undue. These factors include, but are not limited to: 1) nature of the invention, 2) state of the prior art, 3) relative skill of those in the art, 4) level of predictability in the art, 5) existence of working examples, 6) breadth of claims, 7) amount of direction or guidance by the inventor, and 8) quantity of experimentation needed to make or use the invention. *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (FED. Cir. 1988).

In the instant case, the nature of the invention, treatment of neurological diseases and prevention of neuronal degeneration, is complex. The claims are very broad because they are not limited to treatment of a single disease, but rather encompass any sort of neuronal degeneration. Claim 1, for example, encompasses prevention of all neuronal degeneration. Claim 21 does not include prevention, but encompasses all neurodegenerative conditions, disorders, and diseases. Claim 40 encompasses amelioration of neurodegeneration associated with injury, no matter what that injury might be.

The specification discloses several working examples in which Poly-Glu,Tyr was administered to animals with certain very specific injuries. Examples 1 – 2 show that subcutatneous administration of the agent decreases the number of neurons that die within the retina after administration of glutamate. Examples 4 and 12 show that administration of the agent attenuates the severity of certain behavioral sequelae of spinal cord injury and ischemic stroke. Example 6 shows that animals receiving the agent have attenuated cell loss in a chronic model of glaucoma, but Example 7 shows a minimal effect in acute glaucoma, if one is present at all. However none of the examples in the specification show prevention of neurodegeneration, which is recited in claim 1. The art recognizes that neural death, or degeneration, is a normal part of the aging process. See for example the enclosed article by Bussiere et al., which teaches the rates of neurodegeneration in normal human brains and compares it to the rates seen in patients with dementia. Thus all patients are in need of prevention of neurodegeneration. The specification does not show the complete arrest of

degeneration of neurons with advanced age, and thus does not provide enablement for prevention as broadly claimed in claim 1.

The specification also does not provide enablement for promoting or inhibiting nerve regeneration, or the amounts of Poly-Glu,Tyr effective for such. The specification discloses a prophetic example for determining the numbers of new neurons born after ischemic stroke. This is set forth in Example 15, beginning on p. 49. No actual results of experiments are reported. The example discloses that "poly-Glu,Tyr has a positive effect on adult neurogenesis in the brain after ischemia" (p. 50 lines 13 – 14). However the specification does not disclose what constitutes a "positive effect". The clearest definition of "positive" would seem to mean that the agent increases the number of neurons. However, the specification also discloses that neurogenesis is the body's own reaction to pathological insult such as stroke. Thus in the context of the example, "positive effect" seems to be a decrease in the number of neurons born. There are no working examples of changing the numbers of neurons, either increasing or decreasing them, by administering poly-Glu, Tyr. As the specification teaches that the increase in neurogenesis is a consequence of injury, but the art recognizes that neurogenesis is beneficial and correlated with cognitive function (see for example Kemperman 2002. Journal of Neuroscience 22:635-638) , and since there is no disclosure of whether poly-Glu,Tyr increases or decreases neurogenesis, the skilled artisan would essentially have to determine whether the agent increases or decreases neurogenesis, and then determine the dose effective for this function. Given the contradictions between the art, which teaches neurogenesis is beneficial, and the specification which teaches that it is a consequence of injury, it would take undue experimentation by the artisan to practice the method commensurate in scope with the claims.

Furthermore, the specification is not enabling for treatment of all diseases and conditions associated with neural cell death. Many diseases lead to a patient being in need of nerve regeneration but the cell death associated with the disease is not due to glutamate toxicity. The specification discloses a prophetic example of treatment of amyotrophic lateral sclerosis by administration of poly-Glu,Tyr (see p. 52 – 53). However the compound was not actually administered to patients, or even to mice with a reasonable model of the disease. There is no disclosure of amelioration of symptoms, or slowing of disease onset by administering poly-Glu,Tyr. One of skill in the art would not expect the agent to have any effect on Huntington's disease, as the death of neurons is not caused by injury or by excessive glutamate release, which the working examples in the specification point to as conditions suitable for treatment with

the agent. Orr (2001. Genes and Development 15:925-932) teaches that the disease is caused by expansion of polyglutamine repeats in the huntingtin protein, and that this leads to formation of aggregates and subsequent cell death. As the mechanism underlying cell death in Huntington's disease is quite different from that underlying cell death due to excessive glutamate, administration of poly-Glu,Tyr would not be expected to be effective for treatment of Huntington's, or for any disease or condition which does not share a mechanism with those conditions shown to be amenable to treatment. Thus given the state of the art, the breadth of the claims, and the lack of sufficient guidance and working examples in the specification, it would take undue experimentation on the part of a skilled artisan to practice the claimed methods commensurate in scope with the claims.

Claim Rejections - 35 USC § 102

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claim 1 is rejected under 35 U.S.C. 102(b) as being anticipated by Vidovic (1985. Journal of Immunology 134:3563 – 3568, cited on IDS filed 24 March 2004), as evidenced by Bussiere et al. (2001. In Functional Neurobiology of Aging, Hof and Mobbs (Eds.). pp. 77 - 84.).

Claim 1 encompasses prevention of neuronal degeneration by administering poly Glu,Tyr "to an individual in need thereof". Bussiere provides evidence that this includes all subjects, as all subjects lose neurons in advancing age. Hence, all subjects are in need of prevention of neuronal degeneration, as well as inhibition of neuronal degeneration, promotion of nerve regeneration in both the CNS and the PNS. As all nerves are subject to glutamate toxicity, all subjects are in need of protection of nerves from glutamate toxicity. Vidovic teaches administration of poly-Glu,Tyr to mice at a dose of 0.1 mg in 0.1 ml of adjuvant. (see p. 3563, "Antigens and Immunization"). Assuming a mouse weighs 30 g, this comes to a dose of 3.333 mg/kg body weight. The specification discloses that doses of 150 – 1000 ug of poly-Glu,Tyr per rat are effective in attenuating the behavioral effects of stroke. Assuming a rat weighs 300 g, this comes to a dose of 0.5 – 3.333 mg/kg. As the Vidovic reference teaches administration of

the same dose of the same compound to subjects in need of prevention and inhibition of neurodegeneration, and in need of promotion of nerve regeneration in both the CNS and PNS, and in need of protection of nerves from glutamate toxicity, it anticipates the claimed invention.

Claim Rejections - 35 USC § 103

7. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

8. Claims 1 – 3, 21 – 22, 40 – 41, and 45 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kipnis (2000. Proc Natl Acad Sci USA 97:7446-7451, cited on IDS filed 3 August 2004) and Vidovic (1985. Journal of Immunology 134:3563-3568, cited on IDS filed 24 March 2004).

Kipnis teaches administration of copolymer 1 (Cop-1) inhibits the progression of secondary degeneration after nerve crush injury, which is relevant to claims 1 – 3, 21 – 22, and 40 – 41. Kipnis teaches that Cop-1 is a synthetic polymer of four amino acids, alanine, lysine, glutamic acid, and tyrosine (see p. 7446, first column). This protein is similar to, but not the same as, the poly Glu,Tyr recited in all pending claims as the prior art protein has both alanine and lysine, which are not in the protein used in the claimed methods; in the instant case the protein used only has two of the four amino acids used in the protein from Kipnis. Kipnis teaches that immunization with copolymer-1 not only protects neurons within the retina against secondary injury, but Kipnis also teaches that the reason it has these protective effects is that it evokes an immune response. As the neurons in the retina are part of the central nervous

system, the Kipnis reference is clearly on point to inhibition of neuronal degeneration, and promoting nerve regeneration in the CNS, which are recited in generic claim 1. Kipnis also teaches administration of the copolymer to patients suffering from neurologogical injuries, recited in generic claims 21 and 40. The nerve crush injury is a form of a neurological disorder or condition, recited in claim 45. The reference teaches that the random copolymer Cop-1 is non-encephalitogenic (see p. 7450, top of second column) when injected, and elicits T-cell responses. Kipnis teaches that this T-cell response is the mechanism by which the copolymer has its protective effect, and suggests that this mechanism will be useful for protecting CNS neurons from chronic and acute injury (see p. 7451, final paragraph). Furthermore Kipnis teaches that activated T cells are able to pass through the blood-brain barrier (p. 7450, second column), which allows them to have access to the site of injury. While Cop-1 was known to cross-react with myelin basic protein (MBP), Kipnis teaches that this feature is not what is crucial to the protective aspect of T cells, as the Cop-1 administrations do not result in EAE, and the T cells evoked by this drug are regulatory in nature (see p. 7450, last paragraph). Thus the reference teaches the artisan of ordinary skill that proliferation of T cells, by any suitable mechanism, is useful for protecting CNS neurons from damage due to chronic or acute injury. However Kipnis does not teach administration of poly Glu,Tyr.

Vidovic teaches that the random copolymer poly Glu, Tyr (also called poly Glu50Tyr50 to indicate that it is a 100-amino acid protein with Glu and Tyr in an equimolar ratio) induces strong T-cell responses. Vidovic teaches the artisan which mouse strains are particularly strong responders, in terms of how well their T-cells respond to the poly Glu,Tyr injections (see Table 1 on p. 3564). Vidovic teaches that the T-cells are most likely helper-inducers (see p. 3565, second column). The reference fairly teaches proliferation of T cells by administration of poly Glu,Tyr and teaches the amount needed to induce this response and the ways subjects with different genetic backgrounds differ in their proliferative T cell responses. However Vidovic does not teach administration of poly Glu,Tyr for protecting neurons for damage from injury.

It would have been obvious to one of ordinary skill in the art to modify the method of Kipnis et al., which is a method of providing neuronal protection by administering a random copolymer which induces T-cell proliferation, to administer poly Glu,Tyr, which Vidovic teaches is a random copolymer that induces T-cell proliferation, with a reasonable expectation of success. The motivation to do so would be to prevent neuronal damage, which Kipnis teaches is accomplished by administration of the random copolymer that induces T-cell proliferation.

The references provide not only the motivation to select poly Glu,Tyr, but also the reasonable expectation of success, as both references teach that the copolymers induce T-cell proliferation. Thus claims 1 – 3 are obvious over the two references. Claims 21 – 22 differ only in the preamble, which encompass a different intended use. However as the method itself, administration of poly Glu,Tyr for neuroprotection, is the same as in claims 2 – 3, these claims are rejected for the same reason. Similarly claims 40 – 41 also require administration of poly Glu,Tyr for neuroprotection and thus are also rejected.

9. Claims 1 – 4, 6, 10, 21 – 23, 25 – 26, 40 – 42, 44 – 46 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kipnis and Vidovic as applied to claims 1 – 3, 21 – 22, 40 – 41, and 45 above, and further in view of Cecil Textbook of Medicine, 2000, pp. 2092 – 2109)

The reasons why claims 1 – 3, 21 – 22, 40 – 41, and 45 are obvious over Kipnis and Vidovic are set forth in the previous rejection. Briefly, Kipnis teaches that administration of a copolymer which stimulates T-cell proliferation is neuroprotective against secondary neural damage, and teaches administration of this copolymer to patients that fall within the scope of patients recited in all generic claims under examination. Vidovic teaches that a different random copolymer, poly Glu,Tyr, also stimulates T-cell proliferation. However neither Kipnis or Vidovic teaches administration to patients with ischemic stroke or with conditions caused or exacerbated by glutamate toxicity.

Cecil Textbook of Medicine teaches that ischemic stroke is the most common form of stroke, and makes up about 80% of all stroke patients (see p. 2092, first paragraph of Chapter 469). Cecil also teaches that ischemic strokes result in neuronal damage (see p. 2097 bottom of second column), and that such damage often occurs on a time scale considerably longer than that of the initial ischemic event. Particularly, the reference teaches that "the time required for histologic changes to reach their maximum... differs markedly from the time course of injury encountered in selective ischemic necrosis... selective ischemic necrosis of neurons evolves more slowly and sometimes requires several days or more to reach its full extent." (p. 2098, top of second column). Thus Cecil clearly teaches that ischemic stroke is a disease characterized that leads to secondary neural damage. Cecil goes on to say that depolarization of neurons leads to excessively high levels of excitatory neurotransmitters, which can further exacerbate the degree of injury (p. 2098, middle of second column) and particularly points to glutamate as

one of the agents involved in damage. On p. 2108, first column, Cecil teaches that neuroprotective drugs, to be administered for protection against the damage induced by ischemic stroke, currently under investigation include those that block the excessive glutamate release typically seen in stroke. Thus the reference is relevant to claims 4, 6, 10, 23, 25-26, 42, and 44 – 46, drawn to ischemic stroke and conditions characterized by excessive glutamate. However Cecil Textbook of Medicine does not teach administration of poly Glu,Tyr for neuroprotection.

It would have been obvious to one of ordinary skill in the art to administer poly Glu,Tyr for protection in ischemic stroke or in conditions characterized or exacerbated by glutamate toxicity, such as stroke, shy with a reasonable expectation of success. The motivation to do so would be to treat patients with ischemic stroke, offering neuroprotection to them. This motivation flows directly from the prior art references, as Kipnis is on point to neuroprotection, and Cecil Textbook of Medicine teaches that patients with stroke are in need of such neuroprotection, thereby guiding the artisan to this particular patient population for treatment.

Double Patenting

10. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with

this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1 – 4, 6, 10, 21 – 23, 25 – 26, 40 – 42, and 44 – 46 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1 – 6 of U.S. Patent No. 6,835,711. Although the conflicting claims are not identical, they are not patentably distinct from each other because in both cases the claims encompass methods of decreasing damage of certain types of neurons, damaged by or subject to damage by glutamate, by administration of poly-Glu,Tyr.

Conclusion

11. No claim is allowed.

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Daniel Kolker whose telephone number is (571) 272-3181. The examiner can normally be reached on Mon - Fri 8:30AM - 5:00PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Janet Andres can be reached on (571) 272-0867. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

SHARON TURNER, PH.D.
PRIMARY EXAMINER
5-30-06

Daniel E. Kolker, Ph.D.
May 24, 2006